

10

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13sep09 09:07:41 User208760 Session D3109.1
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$0.60 Estimated cost File1
$0.60 Estimated cost this search
$0.60 Estimated total session cost 0.166 DialUnits
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File 410:The Chronolog 2009  
(c) 2009 Dialog. All rights.reserved.

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Set Items Description
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HILIGHT set on as ''
HILIGHT set on as ''
? begin 5,73,155,399
13sep09 09:07:47 User208760 Session D3109.2
$0.00 0.119 DialUnits File410
$0.00 Estimated cost File410
$0.02 TELNET
$0.02 Estimated cost this search
$0.62 Estimated total session cost 0.285 DialUnits
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SYSTEM:OS - DIALOG OneSearch

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File 5:Biosis Previews(R) 1926-2009/Sep W1
(c) 2009 The Thomson Corporation
File 73:EMBASE 1974-2009/Sep 09
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\*File 73: EMBASE Classic available to all Dialog customers.

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File 155:MEDLINE(R) 1950-2009/Sep 11
(c) format only 2009 Dialog
File 399:CA SEARCH(R) 1967-2009/UD=15111
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IPCR/8 classification codes now searchable as IC=. See HELP NEWSIPCR.

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Set Items Description
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Ref Items Index-term
E1 4 AU=FRENTSCH M.
E2 12 AU=FRENTSCH MARCO
E3 0 *AU=FRENTSCH MARCO ?
E4 4 AU=FRENTSCH, MARCO
E5 1 AU=FRENTSEL, I.
E6 1 AU=FRENTSEL, KH.
E7 1 AU=FRENTSEL' G-Y
E8 3 AU=FRENTSEL' KH
E9 1 AU=FRENTSOS J A
E10 1 AU=FRENTSOS J.A.
E11 1 AU=FRENTITE S
E12 2 AU=FRENTZ B
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Enter P or PAGE for more

? s e1-e4

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4 AU=FRENTSCH M.
12 AU=FRENTSCH MARCO
0 AU=FRENTSCH MARCO ?
4 AU=FRENTSCH, MARCO
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S1 20 E1-E4  
 ? e au=rothe martin ?

Ref	Items	Index-term
E1	1	AU=ROTHER MARTIL JILL
E2	4	AU=ROTHER MARTIN
E3	0	*AU=ROTHER MARTIN ?
E4	2	AU=ROTHER MARTINA
E5	3	AU=ROTHER MATTHIAS
E6	3	AU=ROTHER MAURICE
E7	3	AU=ROTHER MEYER A
E8	10	AU=ROTHER MICHAEL
E9	48	AU=ROTHER MIKE
E10	2	AU=ROTHER MIRIAM
E11	5	AU=ROTHER N
E12	1	AU=ROTHER N VINGE

Enter P or PAGE for more

? s e2  
 S2 4 AU='ROTHER MARTIN'  
 ? e au=thiel andreas ?

Ref	Items	Index-term
E1	7	AU=THIEL ANDRA
E2	124	AU=THIEL ANDREAS
E3	0	*AU=THIEL ANDREAS ?
E4	5	AU=THIEL ANDREW J
E5	2	AU=THIEL ANGELA
E6	3	AU=THIEL ANJA
E7	4	AU=THIEL ANNETTE
E8	3	AU=THIEL ANSGAR
E9	6	AU=THIEL AYLIN
E10	67	AU=THIEL B
E11	10	AU=THIEL B A
E12	1	AU=THIEL B D

Enter P or PAGE for more

? s e2  
 S3 124 AU='THIEL ANDREAS'  
 ? s (s1 or s2 or s3) and (cd154 or cd40L or cd40 ligand or gp39) (20n)(block? or suppress? or inhibit? or prevent? or antagoni?) (20n)(diagnos? or detect? or isolat?) (20n)(antigen(w)specific) (20n)(t(w)cell? or t(w)lymphocyt? or cd4? or cd8?)

Processing  
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 Processing  
 Processing  
 Processing

20	S1
4	S2
124	S3
4209	CD154
9464	CD40L
8686	CD40 LIGAND
922	GP39
1763121	BLOCK?
1196060	SUPPRESS?
5634636	INHIBIT?
3137220	PREVENT?
1462371	ANTAGONI?

7027276 DIAGNOS?  
 4015149 DETECT?  
 3352506 ISOLAT?  
 1621823 ANTIGEN  
 4144673 SPECIFIC  
 2389965 T  
 14264231 CELL?  
 884805 T(W)CELL?  
 2389965 T  
 1507666 LYMPHOCYT?  
 579678 T(W)LYMPHOCYT?  
 419460 CD4?  
 228709 CD8?  
 22 (((CD154 OR CD40L) OR CD40 LIGAND) OR  
 GP39) (20N) (((BLOCK? OR SUPPRESS?) OR INHIBIT?) OR  
 PREVENT?) OR ANTAGONI?) (20N) ((DIAGNOS? OR DETECT?) OR  
 ISOLAT?) (20N) ANTIGEN(W) SPECIFIC (20N) ((T(W)CELL? OR  
 T(W)LYMPHOCYT?) OR CD4?) OR CD8?)  
 S4 4 (S1 OR S2 OR S3) AND (CD154 OR CD40L OR CD40 LIGAND OR  
 GP39) (20N) (BLOCK? OR SUPPRESS? OR INHIBIT? OR PREVENT?  
 OR ANTAGONI?) (20N) (DIAGNOS? OR DETECT? OR  
 ISOLAT?) (20N) (ANTIGEN(W) SPECIFIC) (20N) (T(W)CELL? OR  
 T(W)LYMPHOCYT? OR CD4? OR CD8?)

? rd s4

S5 2 RD S4 (unique items)

? t s5/3/all

5/3/1 (Item 1 from file: 5)  
 DIALOG(R)File 5: Biosis Previews(R)  
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0019901393 BIOSIS NO.: 200700561134  
 Identification and isolation of murine antigen-reactive T cells according  
 to CD154 expression  
 AUTHOR: Kirchhoff Dennis; Frentsch Marco; Leclerk Patrick; Bumann Dirk;  
 Rausch Sebastian; Hartmann Susanne; Thiel Andreas; Scheffold  
 Alexander (Reprint)  
 AUTHOR ADDRESS: Deutsches Rheuma Forschungszentrum Berlin, Immunomodulat  
 Grp, Charitepl 1, D-10117 Berlin, Germany\*\*Germany  
 AUTHOR E-MAIL ADDRESS: scheffold@drfz.de  
 JOURNAL: European Journal of Immunology 37 (9): p2370-2377 SEP 2007 2007  
 ITEM IDENTIFIER: doi:10.1002/eji.200737322  
 ISSN: 0014-2980  
 DOCUMENT TYPE: Article  
 RECORD TYPE: Abstract  
 LANGUAGE: English

5/3/2 (Item 2 from file: 5)  
 DIALOG(R)File 5: Biosis Previews(R)  
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17794216 BIOSIS NO.: 200400161557  
 Direct characterisation of adenovirus-specific T helper (Th)-cells in  
 healthy adult donors.  
 AUTHOR: Siegert Stefanie (Reprint); Rescher Ulrike; Chmielewicz Barbara;  
 Frentsch Marco (Reprint); Ellerbrok Heinz; Radbruch Andreas  
 (Reprint); Scheffold Alexander (Reprint); Thiel Andreas (Reprint)  
 AUTHOR ADDRESS: Klinische Immunologie, Deutsches Rheuma-Forschungszentrum,  
 Berlin, Germany\*\*Germany  
 JOURNAL: Blood 102 (11): p53b November 16, 2003 2003

MEDIUM: print  
 CONFERENCE/MEETING: 45th Annual Meeting of the American Society of  
 Hematology San Diego, CA, USA December 06-09, 2003; 20031206  
 SPONSOR: American Society of Hematology  
 ISSN: 0006-4971  
 DOCUMENT TYPE: Meeting; Meeting Abstract  
 RECORD TYPE: Abstract  
 LANGUAGE: English  
 ? s (cd154 or cd40L or cd40 ligand or gp39) (20n)(block? or suppress? or inhibit?  
 or prevent? or antagoni?) (20n)(diagnos? or detect? or  
 isolat?) (20n)(antigen(w)specific) (20n)(t(w)cell? or t(w)lymphocyt? or cd4? or cd8?)  
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4209	CD154
9464	CD40L
8686	CD40 LIGAND
922	GP39
1763121	BLOCK?
1196060	SUPPRESS?
5634636	INHIBIT?
3137220	PREVENT?
1462371	ANTAGONI?
7027276	DIAGNOS?
4015149	DETECT?
3352506	ISOLAT?
1621823	ANTIGEN
4144673	SPECIFIC
2389965	T
14264231	CELL?
884805	T(W)CELL?
2389965	T
1507666	LYMPHOCYT?
579678	T(W)LYMPHOCYT?
419460	CD4?
228709	CD8?
S6	24 (CD154 OR CD40L OR CD40 LIGAND OR GP39) (20N)(BLOCK? OR SUPPRESS? OR INHIBIT? OR PREVENT? OR ANTAGONI?) (20N)(DIAGNOS? OR DETECT? OR ISOLAT?) (20N)(ANTIGEN(W)SPECIFIC) (20N)(T(W)CELL? OR T(W)LYMPHOCYT? OR CD4? OR CD8?)

? rd s6  
 S7 10 RD S6 (unique items)  
 ? t s7/3/all

7/3/1 (Item 1 from file: 5)  
 DIALOG(R)File 5: Biosis Previews(R)  
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0020918436 BIOSIS NO.: 200900258770  
 Disruption of T Cell Suppression in Chronic Lymphocytic Leukemia by CD200  
 Blockade  
 AUTHOR: Pallasch Christian P (Reprint); Ulbrich Susanne; Brinker Reinhild;  
 Uger Robert A; Hallek Michael; Wendtner Clemens-Martin  
 AUTHOR ADDRESS: Univ Cologne, Ctr Integrated Oncol Cologne Bonn, Dept  
 Internal Med 1, Cologne, Germany\*\*Germany  
 JOURNAL: Blood 112 (11): p721-722 NOV 16 2008 2008  
 CONFERENCE/MEETING: 50th Annual Meeting of the American-

Society-of-Hematology San Francisco, CA, USA December 06 -09, 2008;  
20081206  
SPONSOR: Amer Soc Hematol  
ISSN: 0006-4971  
DOCUMENT TYPE: Meeting; Meeting Poster  
RECORD TYPE: Abstract  
LANGUAGE: English

7/3/2 (Item 2 from file: 5)  
DIALOG(R)File 5:BIOSIS Previews(R)  
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0020638860 BIOSIS NO.: 200800685799  
Impact of myelin-specific antigen presenting B cells on T cell activation  
in multiple sclerosis  
AUTHOR: Harp Christopher T; Lovett-Racke Amy E; Racke Michael K; Frohman  
Elliot M; Monson Nancy L (Reprint)  
AUTHOR ADDRESS: Univ Texas SW Med Ctr Dallas, Dept Neurol, Dallas, TX 75390  
USA\*\*USA  
AUTHOR E-MAIL ADDRESS: Nancy.Monson@UTSouthwestern.edu  
JOURNAL: Clinical Immunology (Orlando) 128 (3): p382-391 SEP 2008 2008  
ITEM IDENTIFIER: doi:10.1016/j.clim.2008.05.002  
ISSN: 1521-6616  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

7/3/3 (Item 3 from file: 5)  
DIALOG(R)File 5:BIOSIS Previews(R)  
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0020089819 BIOSIS NO.: 200800136758  
Intracellular CD154 expression reflects antigen-specific CD8(+) T cells but  
shows less sensitivity than intracellular cytokine and MHC tetramer  
staining  
AUTHOR: Han Young Woo; Aleyas Abi G; George Junu A; Yoon Hyun A; Lee John  
Hwa; Kim Byung Sam; Eo Seong Kug (Reprint)  
AUTHOR ADDRESS: Chonbuk Natl Univ, Coll Vet Med, Dept Microbiol, Jeonju  
561756, South Korea\*\*South Korea  
AUTHOR E-MAIL ADDRESS: vetvirus@chonbuk.ac.kr  
JOURNAL: Journal of Microbiology and Biotechnology 17 (12): p1955-1964 DEC  
2007 2007  
ISSN: 1017-7825\_(print) 1738-8872\_(electronic)  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

7/3/4 (Item 4 from file: 5)  
DIALOG(R)File 5:BIOSIS Previews(R)  
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0019901393 BIOSIS NO.: 200700561134  
Identification and isolation of murine antigen-reactive T cells according  
to CD154 expression  
AUTHOR: Kirchhoff Dennis; Frentsch Marco; Leclerk Patrick; Bumann Dirk;  
Rausch Sebastian; Hartmann Susanne; Thiel Andreas; Scheffold Alexander  
(Reprint)  
AUTHOR ADDRESS: Deutsches Rheuma Forschungszentrum Berlin, Immunomodulat

Grp, Charitepl 1, D-10117 Berlin, Germany\*\*Germany  
AUTHOR E-MAIL ADDRESS: scheffold@drfz.de  
JOURNAL: European Journal of Immunology 37 (9): p2370-2377 SEP 2007 2007  
ITEM IDENTIFIER: doi:10.1002/eji.200737322  
ISSN: 0014-2980  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

7/3/5 (Item 5 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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19070219 BIOSIS NO.: 200600415614  
The generation of thymus-independent germinal centers depends on CD40 but not on CD154, the T cell-derived CD40-ligand  
AUTHOR: Gaspal Fabrina M C; McConnell Fiona M; Kim Mi-Yeon; Gray David; Kosco-Vilbois Marie H; Raykundalia Chandra R; Botto Marina; Lane Peter J L (Reprint)  
AUTHOR ADDRESS: Univ Birmingham, MRC, Ctr Immune Regulat, Biomed Res Inst, Vincent Dr, Birmingham B15 2TT, W Midlands, UK\*\*UK  
AUTHOR E-MAIL ADDRESS: p.j.l.lane@bham.ac.uk  
JOURNAL: European Journal of Immunology 36 (7): p1665-1673 JUL 2006 2006  
ISSN: 0014-2980  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

7/3/6 (Item 6 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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18318845 BIOSIS NO.: 200510013345  
Nonreplicating recombinant vaccinia virus expressing CD40 ligand enhances APC capacity to stimulate specific CD4+ and CD8+ T cell responses  
AUTHOR: Feder-Mengus Chantal; Schultz-Thater Elke; Oertli Daniel; Marti Walter R; Heberer Michael; Spagnoli Giulio C; Zajac Paul (Reprint)  
AUTHOR ADDRESS: Univ Basel Hosp, Res Ctr, ICFS, Lab 404, Dept Surg, Oncol Grp, Hebelstr 20, CH-4031 Basel, Switzerland\*\*Switzerland  
AUTHOR E-MAIL ADDRESS: pzajac@uhbs.ch  
JOURNAL: Human Gene Therapy 16 (3): p348-360 MAR 05 2005  
ISSN: 1043-0342  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

7/3/7 (Item 7 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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17847019 BIOSIS NO.: 200400217074  
Blockade of CD40 pathway enhances the induction of immune tolerance by immature dendritic cells genetically modified to express cytotoxic T lymphocyte antigen 4 immunoglobulin.  
AUTHOR: Sun Wenji; Wang Quanxing; Zhang Lihuang; Liu Yushan; Zhang Min; Wang Chunmei; Wang Jianli; Cao Xuetao (Reprint)  
AUTHOR ADDRESS: Institute of Immunology, Zhejiang University, Hangzhou, 310031, China\*\*China

AUTHOR E-MAIL ADDRESS: caoxt@public3.sta.net.cn  
JOURNAL: Transplantation (Hagerstown) 76 (9): p1351-1359 November 15, 2003  
2003  
MEDIUM: print  
ISSN: 0041-1337  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

7/3/8 (Item 8 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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17794216 BIOSIS NO.: 200400161557  
Direct characterisation of adenovirus-specific T helper (Th)-cells in healthy adult donors.  
AUTHOR: Siegert Stefanie (Reprint); Rescher Ulrike; Chmielewicz Barbara; Frentsch Marco (Reprint); Ellerbrok Heinz; Radbruch Andreas (Reprint); Scheffold Alexander (Reprint); Thiel Andreas (Reprint)  
AUTHOR ADDRESS: Klinische Immunologie, Deutsches Rheuma-Forschungszentrum, Berlin, Germany\*\*Germany  
JOURNAL: Blood 102 (11): p53b November 16, 2003 2003  
MEDIUM: print  
CONFERENCE/MEETING: 45th Annual Meeting of the American Society of Hematology San Diego, CA, USA December 06-09, 2003; 20031206  
SPONSOR: American Society of Hematology  
ISSN: 0006-4971  
DOCUMENT TYPE: Meeting; Meeting Abstract  
RECORD TYPE: Abstract  
LANGUAGE: English

7/3/9 (Item 9 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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16548252 BIOSIS NO.: 200200141763  
Normal Th1 development following long-term therapeutic blockade of CD154-CD40 in experimental autoimmune encephalomyelitis  
AUTHOR: Howard Laurence M; Ostrovidov Serge; Smith Cassandra E; Dal Canto Mauro C; Miller Stephen D (Reprint)  
AUTHOR ADDRESS: Department of Microbiology-Immunology, Northwestern University Medical School, 303 East Chicago Avenue, Chicago, IL, 60611, USA\*\*USA  
JOURNAL: Journal of Clinical Investigation 109 (2): p233-241 January, 2002 2002  
MEDIUM: print  
ISSN: 0021-9738  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

7/3/10 (Item 10 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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14284021 BIOSIS NO.: 199800078268  
Blockade of CD40-CD40 ligand pathway induces tolerance in murine contact hypersensitivity

AUTHOR: Tang Aimin; Judge Thomas A; Turka Laurence A (Reprint)  
AUTHOR ADDRESS: Univ. Pa., 901-B Stellar-Chance Build., 422 Curie Blvd.,  
Philadelphia, PA 19104-6100, USA\*\*USA  
JOURNAL: European Journal of Immunology 27 (12): p3143-3150 Dec., 1997  
1997  
MEDIUM: print  
ISSN: 0014-2980  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English  
? t s77/all

7/7/1 (Item 1 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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0020918436 BIOSIS NO.: 200900258770  
Disruption of T Cell Suppression in Chronic Lymphocytic Leukemia by CD200  
Blockade  
AUTHOR: Pallasch Christian P (Reprint); Ulbrich Susanne; Brinker Reinhild;  
Uger Robert A; Hallek Michael; Wendtner Clemens-Martin  
AUTHOR ADDRESS: Univ Cologne, Ctr Integrated Oncol Cologne Bonn, Dept  
Internal Med 1, Cologne, Germany\*\*Germany  
JOURNAL: Blood 112 (11): p721-722 NOV 16 2008 2008  
CONFERENCE/MEETING: 50th Annual Meeting of the American-  
Society-of-Hematology San Francisco, CA, USA December 06 -09, 2008;  
20081206  
SPONSOR: Amer Soc Hematol  
ISSN: 0006-4971  
DOCUMENT TYPE: Meeting; Meeting Poster  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: Suppression of patients' T cells is a key event in CLL pathogenesis and was demonstrated to be mediated by direct cell-cell contact of malignant CLL cells with T-cells. CD200 plays a critical role in regulating the immune system and has been shown to be up-regulated on the surface of different tumors including CLL. In this study we addressed the effects of CD200 over-expression on CLL cells on autologous T cells in a mixed lymphocyte reaction system. We used native and CD40 ligand (CD40L)-stimulated CLL cells as antigen-presenting cells (APCs) to expand autologous T cells of 14 patients T-cell proliferation was analyzed over 3 weeks of in vitro culture. A functional anti-CD200 antibody (IB9) was added to reveal CD200-mediated immunosuppression in the autologous system. Expansion of patient \*\*\*T\*\*\*-cells was assessed by flow cytometry including intracellular staining of FOXP3. Specificity towards CLL-specific antigens was monitored applying fibromodulin derived peptides for detection of specific T-cells by ELISPOT analysis. T-cell proliferation over 3 weeks of in vitro culture was significantly enhanced compared to control cells when using CD40L-stimulated APCs and an anti-CD200 antibody (p=0.0004). CD200 blockade was further shown to stimulate antigen-specific T-cell responses towards the F2 and F4 peptides of the CLL-associated antigen fibromodulin (p=0.04). Finally, the number of \*\*\*CD4\*\*\*+/CD25high/FOXP3+ T cells (T-reg) was significantly decreased in CD200 treated mixed lymphocyte reaction (p=0.04). In summary, CD200 blockade may provide therapeutic benefits in CLL by (I) enhancing T-cell expansion, (II) augmenting an antigen-specific \*\*\*T\*\*\* cell response with (III) suppression of regulatory T cells. CD200 seems to be an important immunosuppressive molecule in CLL: by CD200 blockade immune suppression can be overcome by altering tolerance



to tumor antigens and deregulation of regulatory T cells. This combination of an immune induction paralleled by a disruption of immunosuppressive factors makes anti-CD200 mAb a powerful tool for future treatment of CLL, possibly in combination with other

7/7/2 (Item 2 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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0020638860 BIOSIS NO.: 200800685799  
Impact of myelin-specific antigen presenting B cells on T cell activation in multiple sclerosis  
AUTHOR: Harp Christopher T; Lovett-Racke Amy E; Racke Michael K; Frohman Elliot M; Monson Nancy L (Reprint)  
AUTHOR ADDRESS: Univ Texas SW Med Ctr Dallas, Dept Neurol, Dallas, TX 75390 USA\*\*USA  
AUTHOR E-MAIL ADDRESS: Nancy.Monson@UTSouthwestern.edu  
JOURNAL: Clinical Immunology (Orlando) 128 (3): p382-391 SEP 2008 2008  
ITEM IDENTIFIER: doi:10.1016/j.clim.2008.05.002  
ISSN: 1521-6616  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: The role of B cells in the pathogenesis of Multiple Sclerosis (MS) is incompletely understood. Here we define a possible role for B cells as myelin-specific antigen presenting cells (B-APCs) in MS. Peripheral blood B cells (PBBC) isolated from both MS patients and healthy controls (HC) were activated in vitro with either CD40L/IL-4 or a Class B CpG oligodeoxynucleotide (CpG CDN)/IL-2. Both activation techniques induced PBBCs to upregulate CD80 and HLA-DR, rendering them more efficient APCs than resting B cells. Although the \*\*\*CD40L\*\*\* /IL-4 B-APCs were highly effective in eliciting CNS-antigen specific proliferation by autologous T-cells, CpG ODN/IL-2 stimulated B cells were not. Furthermore, \*\*\*CD40L\*\*\* /IL-4 B-APC induced responses by autologous CD4(+) T cells were susceptible to blocking with anti-HLA-DR antibody, suggesting that T cell responses were specific for antigen presentation by B-APC. CNS-antigen specific CD8(+) T cell proliferation was also blocked by HLA-DR, suggesting that CD8(+) proliferation is in part dependent on \*\*\*CD4\*\*\* (+) help. (C) 2008 Elsevier Inc. All rights reserved.

7/7/3 (Item 3 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
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0020089819 BIOSIS NO.: 200800136758  
Intracellular CD154 expression reflects antigen-specific CD8(+) T cells but shows less sensitivity than intracellular cytokine and MHC tetramer staining  
AUTHOR: Han Young Woo; Aleyas Abi G; George Junu A; Yoon Hyun A; Lee John Hwa; Kim Byung Sam; Eo Seong Kug (Reprint)  
AUTHOR ADDRESS: Chonbuk Natl Univ, Coll Vet Med, Dept Microbiol, Jeonju 561756, South Korea\*\*South Korea  
AUTHOR E-MAIL ADDRESS: vetvirus@chonbuk.ac.kr  
JOURNAL: Journal of Microbiology and Biotechnology 17 (12): p1955-1964 DEC 2007 2007  
ISSN: 1017-7825\_(print) 1738-8872\_(electronic)

DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

**ABSTRACT:** A recent report showed that analysis of CD 154 expression in the presence of the secretion inhibitor Brefeldin A (Bref A) could be used to assess the entire repertoire of antigen-specific CD4(+) T helper cells. However, the capacity of intracellular CD 154 expression to identify antigen-specific CD8(+) T cells has yet to be investigated. In this study, we compared the ability of intracellular CD154 expression to assess antigen-specific CD8(+) T cells with that of accepted standard assays, namely intracellular cytokine IFN-gamma staining (ICS) and MHC class I tetramer staining. The detection of intracellular CD154 molecules in the presence of Bref A reflected the kinetic trend of antigen-specific CD8(+) T cell number, but unfortunately showed less sensitivity than ICS and tetramer staining. However, ICS levels peaked and saturated 8 h after antigenic stimulation in the presence of Bref A and then declined, whereas intracellular CD154 expression peaked by 8 h and maintained the saturated level up to 24 h post-stimulation. Moreover, intracellular CD 154 expression in antigen-specific CD8(+) T cells developed in the absence of CD4(+) T cells changed little, whereas the number of IFN-gamma-producing CD8(+) T \*\*\*cells\*\*\* decreased abruptly. These results suggest that intracellular CD154 could aid the assessment of antigen-specific CD8(+) T cells, but does not have as much ability to identify heterogeneous \*\*\*CD4\*\*\* (+) T helper cells. Therefore, the combined analytical techniques of ICS and tetramer staining together with intracellular CD154 assays may be able to provide useful information on the accurate phenotype and functionality of antigen-specific CD8(+) T \*\*\*cells\*\*\* .

7/7/4 (Item 4 from file: 5)  
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0019901393 BIOSIS NO.: 200700561134  
Identification and isolation of murine antigen-reactive T cells according to CD154 expression  
AUTHOR: Kirchhoff Dennis; Frentsch Marco; Leclerk Patrick; Bumann Dirk; Rausch Sebastian; Hartmann Susanne; Thiel Andreas; Scheffold Alexander (Reprint)  
AUTHOR ADDRESS: Deutsches Rheuma Forschungszentrum Berlin, Immunomodulat Grp, Charitepl 1, D-10117 Berlin, Germany\*\*Germany  
AUTHOR E-MAIL ADDRESS: scheffold@drfz.de  
JOURNAL: European Journal of Immunology 37 (9): p2370-2377 SEP 2007 2007  
ITEM IDENTIFIER: doi:10.1002/eji.200737322  
ISSN: 0014-2980  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

**ABSTRACT:** T helper (Th) cells are central regulators of adaptive immune responses. However, the \*\*\*detection\*\*\* of the small number of Th cells specific for a particular antigen or pathogen is still a major challenge. CD154 was recently introduced as a marker for antigen-specific Th cells. To date, this technology was not applicable for mice - arguably the most important immunological model system. CD154 is difficult to detect due to its rapid removal from the cell surface upon binding to \*\*\*CD40\*\*\* during \*\*\*antigen\*\*\* - \*\*\*specific\*\*\* activation by APC. We present an efficient strategy to block the degradation of murine CD154 by

combined use of antibodies against \*\*\*CD40\*\*\* and CD154. This strategy makes CD154 easily accessible for surface staining, which allows isolation and expansion of rare \*\*\*antigen\*\*\* \*\*\*specific\*\*\* Tcells. Importantly, CD154 identified all specific Tcells in strongly Th1- or Th2-polarized immune responses against pathogens like Salmonella typhimurium and Heligmosomoides polygyrus, independent of their potential to produce cytokines. We demonstrate that \*\*\*CD154\*\*\* can in fact be used as a reliable marker for antigen-specific CD4 T cells in mice, offering a unique option to analyze, isolate and rapidly expand the entire pool of Th-cells generated during a physiological \*\*\*T\*\*\* \*\*\*cell\*\*\* response in vivo.

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19070219 BIOSIS NO.: 200600415614  
The generation of thymus-independent germinal centers depends on CD40 but not on CD154, the T cell-derived CD40-ligand  
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ABSTRACT: In this report, we show that the formation of germinal center (GC)-like structures to thymus-independent type 2 antigens in mice depends on intact signals through CD40, but does not depend on T cell-derived CD40-ligand (CD 154). In addition, we show that follicular dendritic cells (FDC) are also critical to thymus-independent GC formation, as their depletion by blockade of lymphotoxin-P receptor signals abrogated GC development unless the responding B cells bound antigen with high affinity. Further evidence that immune complexes drove this CD40-dependent B cell proliferation was provided by the observation that an antibody that detects immune complexes containing complement component 4 on FDC also inhibited thymus-independent GC formation when injected in vivo at the time of immunization. Finally, we show that thymus-independent B cell proliferation was associated with class switching to IgG3, as IgG3(+) antigen-specific switched B cells could be visualized directly in GC, suggesting that immune complexes can provide the signals for class switching within GC in the absence of \*\*\*CD154\*\*\*.

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18318845 BIOSIS NO.: 200510013345  
Nonreplicating recombinant vaccinia virus expressing CD40 ligand enhances APC capacity to stimulate specific CD4+ and CD8+ T cell responses  
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**ABSTRACT:** Recombinant poxviruses expressing immunomodulatory molecules together with specific antigens represent powerful vaccines for cancer immunotherapy. Recently, we and others have demonstrated, in vitro and in vivo, that coexpression of CD80 and CD86 costimulatory molecules enhances the immunogenic capacity of a recombinant vaccinia virus (rVV) encoding different tumor-associated antigens. To further investigate the capacity of these vectors to provide ligands for different costimulatory pathways relevant in the generation of T cell responses, we constructed a recombinant virus (rVV) expressing CD40 ligand or CD154 (CD154rVV). Upon binding the CD40 receptor expressed on antigen presenting cells (APC), this molecule, physiologically expressed on activated CD4(+) T cells, increases their antigen presentation and immunostimulatory capacities. Therefore, we evaluated the effects of CD154rVV infection on APC activation and its consequences on T cell stimulation. CD154rVV infection of autologous fibroblasts, monocytes, or iDC promoted the expression of a number of cytokines, including GM-CSF, TNF-alpha, and IL-15 in iDC. Most importantly, IL-12 p40 gene expression and protein secretion were induced by CD154rVV but not by wild-type VV (WT VV) in either CD14(+) cells or iDC, and these effects could be blocked by anti-CD40 monoclonal antibodies. Furthermore, phenotypic characterization of CD154rVV infected iDC revealed enhanced expression of CD83 and CD86 surface markers as compared with wild-type vaccinia virus infection. As expected, VV infection triggered cytokines gene expression in cultures including APC and T cells from VV immune donors. However, cytokine genes typically expressed by T cell receptor triggered T cells such as those encoding IL-2 and IFN-gamma, or T cell proliferation, were detectable to a significantly higher extent in CD154rVV infected cultures, as compared with WT VV. Activation of specific \*\*\*CD8\*\*\* (+) T cells was then investigated using MART-1/Melan-A(27-35) epitope as the model of tumor-associated antigen (TAA). In the presence of CD154rVV activated APCs, significantly higher numbers of specific cytotoxic CD8(+) T cells were detected, as compared with cultures performed in the presence of WT VV or in the absence of virus. Taken together, these data indicate that functional CD154 expression from rVV infected cells promotes APC activation, thereby enhancing antigen-specific T cell generation. Such a recombinant vector might help bypass the requirement for activated helper cells during CTL priming, thus qualifying as a potentially relevant vector in the generation of CD8(+) T \*\*\*cell\*\*\* responses in cancer immunotherapy.

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17847019 BIOSIS NO.: 200400217074

Blockade of CD40 pathway enhances the induction of immune tolerance by immature dendritic cells genetically modified to express cytotoxic T lymphocyte antigen 4 immunoglobulin.

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**ABSTRACT:** Background. Immature dendritic cells (DCs) have the tolerogenic potential to induce alloantigen-specific immune tolerance. Cytotoxic T lymphocyte antigen 4 immunoglobulin (CTLA4Ig) gene-modified immature DCs have been shown to maintain their tolerogenicity and prolong allograft survival to some extent. We investigated whether blockade of CD40 pathway by anti-CD40 ligand (L) monoclonal antibody (mAb) could enhance the immune tolerance induction by immature DCs genetically modified to express CTLA4Ig (DC-CTLA4Ig). Methods. The tolerogenic properties of DC-CTLA4Ig were analyzed. In the vascularized heterotopic heart transplantation murine model, 2X106 DC-CTLA4Ig were infused intravenously into recipients, with or without a concomitant administration of anti-CD40L mAb 7 days before transplantation. Host responses to donor alloantigen were quantified by mixed leukocyte reaction and CTL assays. Donor major histocompatibility complex class II (Iab) expression in recipient lymph nodes was detected posttransplantation by semiquantitative reverse transcriptase-polymerase chain reaction. Results. The allostimulatory activity of DC-CTLA4Ig was reduced. DC-CTLA4Ig also induced alloantigen-specific T-cell hyporesponsiveness and polarized T helper 2 cytokine production. Pretreatment of the recipients with DC-CTLA4Ig modestly prolonged allograft survival, without long-term allograft acceptance. Combined administration of DC-CTLA4Ig and anti-CD40L mAb significantly prolonged cardiac allograft survival, with long-term (> 100 days) survival of 50% of the allografts in the pretreated recipients. More potent donor-specific \*\*\*inhibition\*\*\* of immune response against alloantigens and increased microchimerism were observed in these recipients. Conclusions. \*\*\*Blockade\*\*\* of \*\*\*CD40\*\*\* pathway with anti-CD40L mAb potentiates the tolerogenic potential of DC-CTLA4Ig and enhances the induction of antigen-specific immune tolerance more effectively.

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17794216 BIOSIS NO.: 200400161557  
Direct characterisation of adenovirus-specific T helper (Th)-cells in healthy adult donors.  
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LANGUAGE: English

**ABSTRACT:** Background and Aims: Adenovirus (Ad) infections have been increasingly recognised as an important cause of morbidity and mortality in allogeneic stem cell transplant recipients, especially in children. Immune responses to Ad infection are not fully understood, but T-cell mediated immunity appears to be important for recovery. The target proteins of Ad and their epitopes are still unknown. The aim of the present study was to evaluate and characterise Ad-specific T-cell responses in healthy adult donors. Methods: T cell responses to different Ad serotypes were investigated in healthy adult donors using a short-term stimulation assay. Whole blood was stimulated for 6 hours with anti-CD28 and different antigens (e.g. Ad2, 3, 4, 7, 12 lysates, control lysates, SEB, CMV lysate) in the presence of the secretion inhibitor Brefeldin A. After stimulation, cells were stained for CD69, CD4 and TNF-alpha for FACS analysis. In order to assess the complete fraction of Ad-specific CD4+ Th-cells we also tested the expression of antigen-reactive CD154 (CD40L), as a marker for antigen-specific Th-cells while antigen-reactive TNF-alpha was used to evaluate proinflammatory Th-cells. Results: Ad-specific T cells reactive to at least one of the used lysates could be detected in all of the adult donors analysed. In response to Ad3 lysate 0.06% of CD4+ T-cells became CD69+/TNF-alpha+ (median; range 0.03-0.29%; n=13) compared with 0.01% for anti-CD28 alone or control lysate. Analysis of Ad3-reactive CD154+ expression always revealed slightly higher percentages as compared to TNF-alpha+ expression (0.1%; 0.04-0.79% vs. 0.06%; 0.03-0.29%, n=13). This indicates the feasibility of antigen-reactive CD154 expression after short-term in vitro stimulation for the assessment of the entire repertoire of Th-cells specific for a particular antigen or mixtures of antigens. Of interest, the frequency of specific T cells varied from donor to donor. For example, Ad7 lysate induced strong \*\*\*CD4\*\*\* + Th-cell responses only in selected donors. In one particular healthy donor nearly 5 in 1,000 CD4+ Th-cells (CD4+/CD69+/TNF-alpha+; 0.45% vs. 0.05% control lysate) reacted upon stimulation with Ad7. Conclusions: Our results confirm the previous assumption that Ad-specific Th-cells in human adults may be characterised by widespread crossreactivities for different Ad serotypes. However, since distinct Ad serotypes elicit immune responses only in selected adult donors further analysis of the fine specificities of these Th-cell responses are necessary. Moreover, this will be a prerequisite in order to define protective Ad-specific T-cell responses to develop protocols for specific adoptive immunotherapies in allogeneic stem cell transplantation.

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Normal Th1 development following long-term therapeutic blockade of CD154-CD40 in experimental autoimmune encephalomyelitis

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**ABSTRACT:** Experimental autoimmune encephalomyelitis (EAE) is a Th1-mediated demyelinating disease of the CNS with similarities to multiple sclerosis. We and others have shown that a short-term course of anti-CD154 mAb treatment to block CD154-CD40 interactions can be used to prevent or even treat ongoing PLP139-151-induced relapsing EAE. However, little is known of the long-term effects of CD154 blockade on the development of antigen-specific T cell function. Here, we show that short-term treatment with anti-CD154 at the time of PLP139-151/CFA immunization inhibits clinical disease for up to 100 days after immunization. At this point, comparable numbers of Th1 cells are observed in anti-CD154 and control Ig-treated mice, as assessed by antigen-specific ELISPOT assays. Thus, the long-term Th1/Th2 balance is largely unaffected. Inflammatory responses are diminished in anti-CD154-treated mice, as indicated by reduced in vivo delayed-type hypersensitivity and reduced levels of splenic IFN-gamma secretion in vitro. However, upon adoptive transfer of T cells isolated from the spleens of anti-CD154-treated mice, these cells contributed as effectively to clinical disease as those obtained from control-treated mice. Thus, anti-CD154 therapy leads to long-term therapeutic efficacy without exerting a long-term influence on Th1 development.

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14284021 BIOSIS NO.: 199800078268  
Blockade of CD40-CD40 ligand pathway induces tolerance in murine contact hypersensitivity  
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1997  
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**ABSTRACT:** Interactions between CD40 on antigen-presenting cells and its ligand (CD40L) on T cells has been implicated in T cell-mediated immune responses. Previously, we have shown that contact hypersensitivity (CHS), a cell-mediated cutaneous immune response in reaction to haptens, could be subclassified based on whether the hapten primed for Th1 or Th2 cytokines in cells isolated from draining lymph nodes. We also found that tolerance to a Th2-priming hapten could be induced only by simultaneous blockade of the CD40-CD40L and B7-CD28 at the time of sensitization. Here we demonstrate that blockade of CD40-CD40L signaling alone induces long-lasting unresponsiveness to the Th1 hapten 2,4-dinitrofluorobenzene (DNFB), and inhibits antigen-specific T cell proliferation in vitro. We find that CD40-CD40L signaling is required in the sensitization but not elicitation phase of DNFB-induced CHS, as treatment of mice with anti-CD40L monoclonal antibody (mAb) does not affect the response to hapten challenge in previously sensitized and untreated animals. Examination of cytokine production shows that anti-CD40L mAb decreases interferon-gamma production by draining lymph node cells from

DNFB-sensitized mice, and reciprocally increases interleukin (IL)-4 production. Consistent with this Th1 to Th2 immune deviation, anti-CD40L mAb prevents the induction of IL-12 mRNA in regional lymph nodes, an event which is normally seen within 12 h following hapten sensitization. In contrast, suppression of CHS by CTLA4Ig decreased the production of all cytokines by draining lymph node cells. Together, these data show that blockade of the CD40-CD40L pathway by itself is sufficient to induce tolerance to DNFB-induced CHS, and that this is associated with  
\*\*\*blockade\*\*\* of IL-12 induction and Th1 to Th2 immune deviation.

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